

Sensory Conduction Parameters in Hypothyroidism: A Cross Sectional Study

Ahmad Faraz¹, Sangeeta Singhal¹, M. Mobarak Hossain¹, S. S. Siddiqui²

¹Department of Physiology, J.N. Medical College, Aligarh Muslim University, Aligarh.

²Rajiv Gandhi Centre of Diabetes and Endocrinology, Aligarh Muslim University, Aligarh.

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ABSTRACT

Background: Thyroid hormones play critical roles in differentiation, growth, and metabolism. Thyroid hormone play important role in development of Central Nervous System and in myelination of neurons. hypothyroidism is a chronic and insidious disease caused by failure of thyroid hormone production mostly due to inflammation, thyroidectomy, use of I131, and anti-hyperthyroidism drugs. Involvement of Peripheral nerve is frequent in hypothyroid patients. Objective of study: To evaluate the dysfunction of Sensory Nerve in Hypothyroid patients. **Methods:** 30 patients of hypothyroidism were included in the study, detailed history and clinical examination was performed. 30 normal subjects were included as control. Nerve Conduction Studies were performed in neurophysiology lab of Physiology dept of JNMC Aligarh. **Results:** While comparing the Sensory Nerve Conduction velocity of Median nerves between the two groups, the Sensory NCV of right Median (44.59 ± 8.39) is found to be significantly decreased in hypothyroid subjects. While comparing the Sensory latency of Median nerve between the two groups, the latency of right median nerve ($3.69\pm.69$), left median nerve ($3.43\pm.80$) is significantly increased bilaterally in hypothyroid subjects. **Conclusion:** Hypothyroidism is a definite cause of neuropathy, it affects sensory nerves first than motor nerves. We suggest performing electrophysiological studies in hypothyroid patients, even asymptomatic patients in an early course of disease. So we can prevent the progression of neuropathy.

Keywords: Hypothyroidism, Thyroid Hormone.

INTRODUCTION

Thyroid hormones play critical roles in differentiation, growth, and metabolism. Thyroid hormone play important role in development of Central Nervous System and in myelination of neurons. Thyroid hormone has major effects on the developing brain in utero and during the neonatal period.^[1] Hypothyroidism is a chronic and insidious disease caused by failure of thyroid hormone production. Hypothyroidism is one of the major causes of dysfunction of peripheral nerves, carpal tunnel syndrome is major presentation of mononeuropathy. Approximately one third of patients develop proximal upper and lower limbs muscle weakness, fatigue, myalgia, and muscle cramps.^[2]

The most common entrapment neuropathy is the carpal tunnel syndrome (CTS), compression of median nerve at the wrist, due to the accumulation of amyloglycane matter.^[3] Neuromuscular derangements in hypothyroid patients are mainly metabolic. Main metabolic derangements are the

reduction of fatty acid and carbohydrate metabolism which leads to diminished production of ATP.

Adrenergic receptors on muscle cells are also reduced in hypothyroid patients resulting in diminished glycogenolysis. Deposits of mucopolysaccharide in peripheral nerves and metabolic disorders of Schwann cells may contribute to peripheral nerve involvement in hypothyroid patients.^[4]

MATERIALS AND METHODS

Cases:

Thirty patients of Hypothyroidism with increased serum Thyroid Stimulating Hormone (TSH), and decreased serum FT4 were taken from Rajiv Gandhi Centre of Diabetes and Endocrinology. Written and Informed Consent were taken from all the cases that were included in the study. Patient with overt hypothyroidism were included in the study. Patients having Diabetes Mellitus, Hypertension, vitamin B12 deficiency and history of Neuropathy, patients taking steroids, neuropathy from other conditions were excluded from the study.

Control:

Thirty normal subjects who gave informed consent and are willing were included in our study.

Name & Address of Corresponding Author

Dr Ahmad Faraz,
Department of Physiology,
J.N. Medical College,
Aligarh Muslim University,
Aligarh.

Clinical Evaluation:

Patients included in the study were screened for neuromuscular symptoms including weakness, fatigue, myalgia, stiffness, cramps, numbness, tingling, pain, and paresthesia. Neurologic examination for motor and sensory were done. Sensory examination consisted of pin-prick, light touch, and vibration sensation

Nerve Conduction Studies: NCS were carried out according to standardized protocols for bilateral median, ulnar, and sural sensory response. Antidromic recording was done in the present study. In antidromic conduction study, the nerve is stimulated at a proximal point and SNAP is recorded distally. Latencies and Nerve Conduction Velocity were recorded. Abnormalities were defined as deviation from reference values for the testing neurophysiology laboratory, derived from standard norms in the medical literature.^[5] The increased SD and decreased SNCV in any nerve indicate sensory conduction impairment of that nerve.

Statistical analyses:

Were done using SPSS 21.0. Normalcy of data was checked by Kolmogrov Smirnoff test, Data were expressed as mean \pm SD. Unpaired t-tests were used for comparison between quantitative variables. Chi-square tests were used to compare differences in the categorical variables. Assessment of correlation between variables was performed using Pearson coefficients. The confidence interval (CI) was set at 95%.

RESULTS

While comparing the SNCV between the two groups, the SNCV of right median (44.59 ± 8.39) is found to be significantly decreased in hypothyroid subjects as compared to the SNCV of right median (51.59 ± 8.40) in the control subjects. While comparing the Sensory latencies between the two groups, the Sensory latency of right median ($3.69 \pm .69$), left median ($3.43 \pm .80$), is found to be significantly increased bilaterally in hypothyroid subjects as compared to the Sensory latency of right median ($3.14 \pm .56$), left median ($3.03 \pm .43$), in the control subjects.

Table 1: Comparison between Sensory Nerve Conduction Velocity (SNCV) of various nerves in both the Hypothyroid (n=30) and the control subjects (n=30) on Right side

| Parameter | Control (n=30) M \pm SD (m/s) | Case (Hypothyroid) (n=30) M \pm SD (m/s) | p Value |
|-------------|------------------------------------|---|---------|
| Median SNCV | 51.59(8.40) | 44.59(8.39) | <.05* |
| Ulnar SNCV | 54.17(8.21) | 52.82(7.84) | >.05 |
| Sural SNCV | 43.87(6.54) | 44.22(5.69) | >.05 |

* P value <.05: Statistically Significant

Table 2: Comparison between Sensory Nerve Conduction Velocity (SNCV) of various nerves in both the Hypothyroid (n=30) and the control subjects (n=30) on Left side

| Parameter | Control (N=30) M \pm SD (M/S) | Case (Hypothyroid) (N=30) M \pm SD (M/S) | P Value |
|-------------|------------------------------------|---|---------|
| Median SNCV | 50.59(8.40) | 48.56(7.20) | >.05 |
| Ulnar SNCV | 56.82(6.54) | 53.95(6.25) | >.05 |
| Sural SNCV | 43.60(6.43) | 44.29(5.58) | >.05 |

* P value <.05: Statistically Significant

Table 3: Comparison between Sensory Nerve Latencies of various nerves in both the Hyperthyroid (n=30) and the control subjects (n=30) on Right side

| Parameter | Control (n=30) M \pm SD (m/s) | Case (Hypothyroid) (n=30) M \pm SD (m/s) | p VALUE |
|----------------|------------------------------------|---|---------|
| Median Latency | 3.14(.56) | 3.69(.69) | <.05* |
| Ulnar Latency | 2.85(.39) | 2.83(.64) | >.05 |
| Sural Latency | 2.81(.45) | 2.95(.54) | >.05 |

* P value <.05: Statistically Significant

Table 4: Comparison between Sensory Nerve Latencies of various nerves in both the Hyperthyroid (n=30) and the control subjects (n=30) on Left side

| Parameter | Control (n=30) M \pm SD (m/s) | Case (Hypothyroid) (n=30) M \pm SD (m/s) | p Value |
|----------------|------------------------------------|---|---------|
| Median Latency | 3.03(.43) | 3.43(.80) | <.05* |
| Ulnar Latency | 2.96(.50) | 3.04(.38) | >.05 |
| Sural Latency | 2.75(.37) | 2.85(.39) | >.05 |

* P value <.05: Statistically Significant

DISCUSSION

In present study sensory neuropathy was present in Median nerve as latency were increased and velocity was decreased. Study conducted by Eslamian et al also found similar result.^[6] El salem et al also found similar result.^[7] In our previous study we found derangements in motor nerves as well.^[8] Hypothyroid patient presents with weight gain which might be due to accumulation of mucopolysaccharides in the interstitial spaces which, retain water along with them resulting in weight gain.^[9] Thyroid hormone seems to increase ATPase activity and, thus the increase in activity of ATP dependent Na⁺/K⁺ pump. The increase in ATPase activity would be associated with an increase of ATP transport through the mitochondrial membranes. In hypothyroidism, the ATP deficiency and the reduced activity of the ATPase enzyme induces decrease in Na⁺/K⁺ pump activity, with consequent alterations of pump dependent axonal transport.^[10] Deposition of mucopolysaccharides in the tissues surrounding the nerves or the myxedematous tissue may also lead to compression over the peripheral nerves there by resulting in swelling and degeneration of the nerves.^[11]

Hypothyroidism causes sensory neuropathy by affecting different peripheral nerves but more commonly the median nerve. Sensory neuropathy is frequent in early stage of disease and sensory nerves are affected earlier than motor nerves. Common complaints are usually pain, cramps, parasthesia of fingers and limbs. So, this group of patients is usually manifested by the features of Carpal Tunnel Syndrome.

Mechanism proposed for neuropathy in hypothyroidism: one is secondary to compression caused by deposition of mucopolysaccharide or mucinous deposits in the soft tissues surrounding peripheral nerves, second is polyneuropathy due to either a demyelinating process or primary axonal degeneration.^[10,12] Dysfunction and abnormalities of myelin sheath of axons and oligodendroglial processes may also be responsible for neuropathy in patients with hypothyroidism.^[13] Some investigators suggested that the weight gain in the hypothyroids may be a contributory factor for neuropathy.

CONCLUSION

We conclude that hypothyroidism causes sensory neuropathy early in the disease state. There is significant decrease in NCV as well as increase in latency, and this decrease in NCV is more prominent in median nerve. We suggest performing electrophysiological studies in hypothyroid patients, even asymptomatic patients in an early course of disease. So we can prevent the progression of neuropathy.

REFERENCES

1. Bernal J. Iodine and brain development (1999). *Biofactors* 10: 271–276.
2. Dumitru D, Amato AA. Acquired neuropathies In: Dumitru D, Amato AA, Zwarts MJ, eds. *Electrodiagnostic medicine*. 2nd ed. Philadelphia, PA: Hanley & Belfus Inc; 2002b:947e948, 984.
3. Khedr EM, El-toony LF, Tarkhan MN, Abdalla G. Peripheral and central nervous system alterations in hypothyroidism: Electrophysiological findings. *neuropsychobiology*, 41(2): 88-94; 2000.
4. Shirabe T, Tawara S, Terao A, Araki S. Myxedematous poly neuropathy. A light and electron microscopy study of the peripheral nerve and muscle. *J. Neurol. Neurosurg. Psychiatry* 1975; 38: 241-7.
5. Mishra UK, Kalita J. *Clinical neurophysiology*. Elsiver pub. 2nd edition 2006.
6. Eslamian, F., Bahrami, A., & Behkamrad, K. (2011). Electrophysiologic Changes in Patients With Untreated Primary. *J Clin Neurophysiol* 2011;28: 323-328.
7. El-Salem K, Ammari F. Neurophysiological changes in neurologically asymptomatic hypothyroid patients: A prospective cohort study. *J Clin Neurophysiol* 2006;23:568-72.
8. Faraz A, Singhal S, Hossain MM, Siddiqui SS. Effect of Hypothyroidism on Motor Nerve Conduction Studies: A Cross Sectional Study. *Ann. Int. Med. Den. Res.* 2017; 3(2):PH01-PH04.
9. Yeasmin S, Begum N, Begum S. Motor neuropathy in hypothyroidism: clinical and electrophysiological findings. *BSMMUJ*. 2008; 1(1): 15–18.
10. Nemni R, Bottachi E, Fazio R. Polyneuropathy in hypothyroidism: clinical, electrophysiological and morphological finding in our cases. *J Neurol Neurosurg Psychiatry* 1987; 50: 1454-60.
11. Jalilzadeh SH, Bahrami A, Eftekharsadat B, et al. Peripheral nerve function in subclinical hypothyroidism: A case-control study. *Int J Endocrinolmetab*. 2006; 4: 78–83.
12. Kececi H, Degirmenci Y. Hormone replacement therapy in hypothyroidism and nerve conduction study. *Clinical Neurophysiology*; 2006; 36: 79-83.
13. Ferracci F, Carnevale A. The neurological disorder associated with thyroid autoimmunity. *J. Neurol*. 2006; 253(8): 975-984.

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